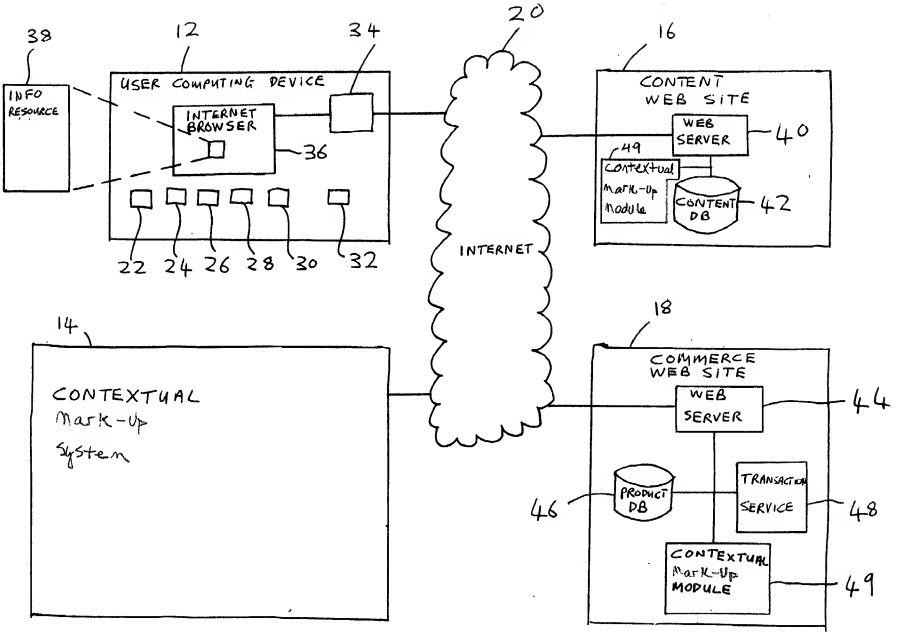
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INFO RESOURCE
12
USER COMPUTING DEVICE
INTERNET BROWSER
36
34
20
16
CONTENT WEB SITE
WEB SERVER
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49
Contextual Mark-Up Module
CONTENT DB
42
22
24
26
28
30
32
INTERNET
14
CONTEXTUAL Mark-Up System
18
COMMERCE WEB SITE
WEB SERVER
44
PRODUCT DB
46
TRANSACTION SERVICE
48
CONTEXTUAL Mark-Up MODULE
49

FIGURE 1

10

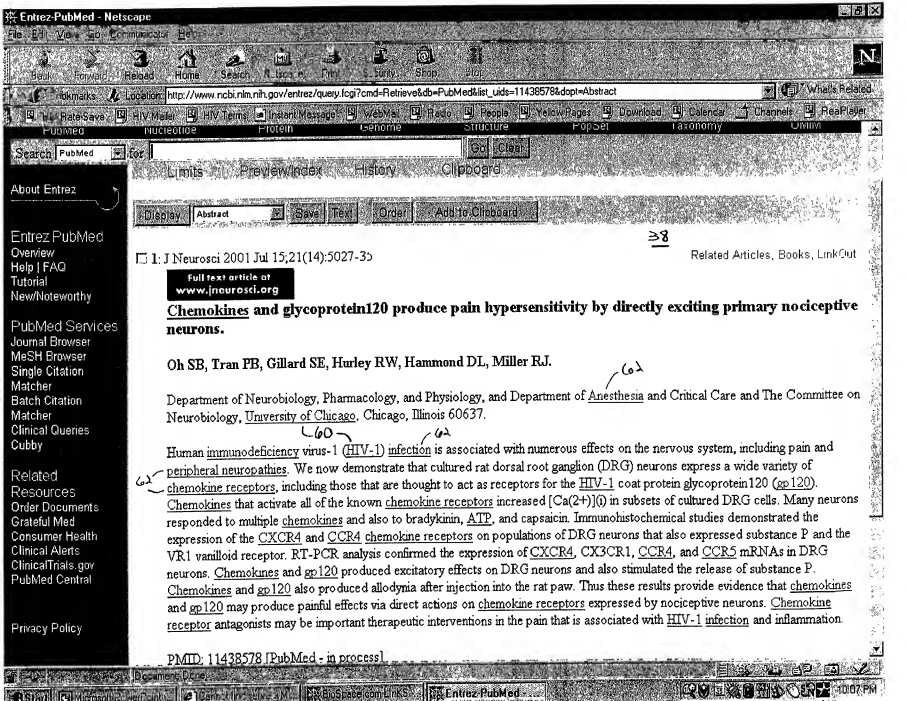
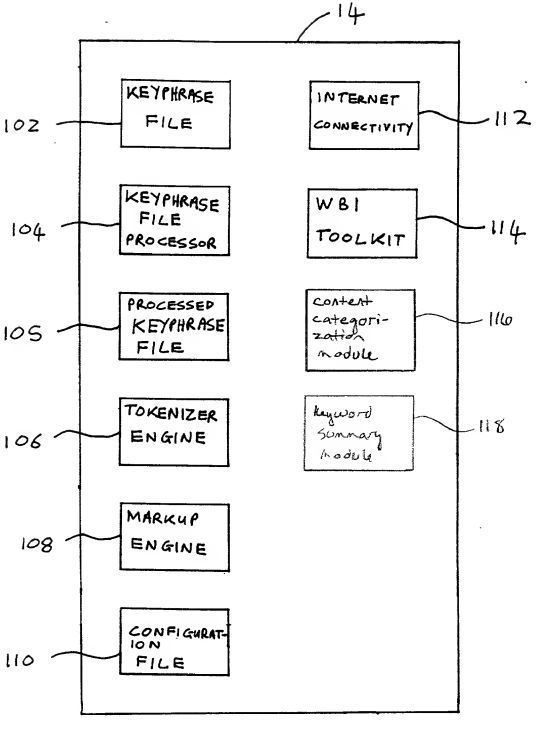


Figure 2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KEYPHRASE FILE
102
KEYPHRASE FILE PROCESSOR
104
PROCESSED KEYPHRASE FILE
105
TOKENIZER ENGINE
106
MARKUP ENGINE
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CONFIGURATION FILE
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INTERNET CONNECTIVITY
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WBI TOOLKIT
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content categorization module
116
keyword summary module
118

FIG. 3

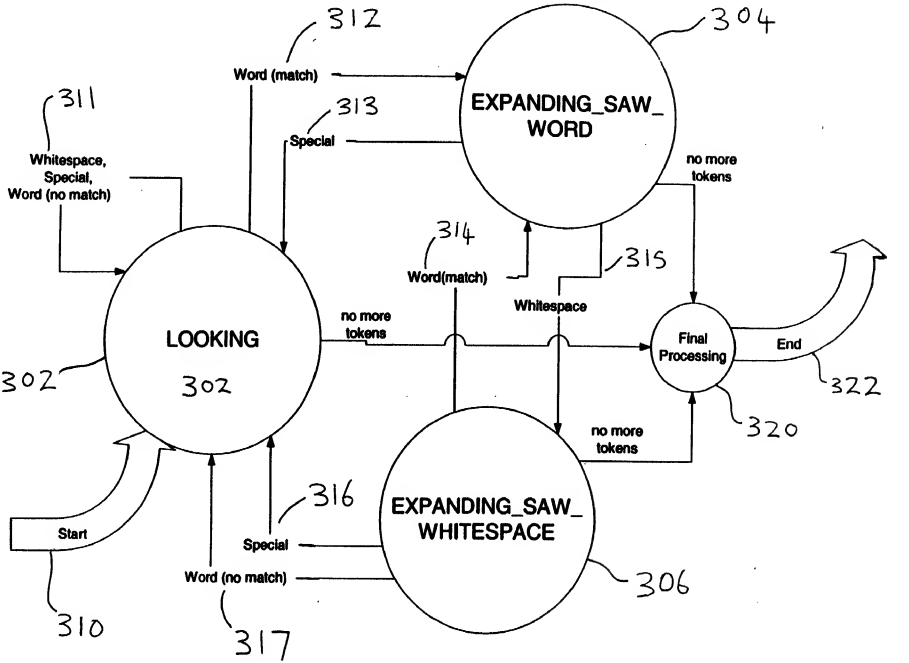


FIGURE 4

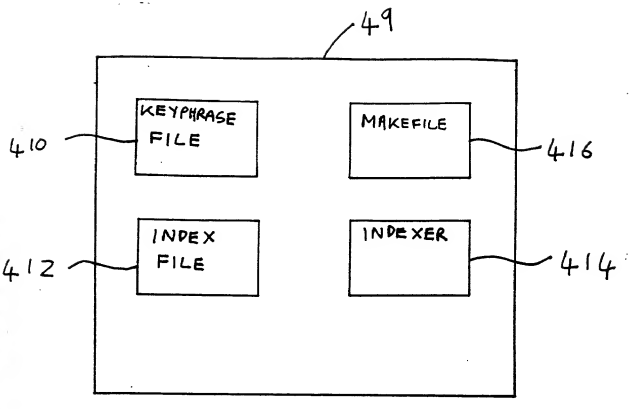
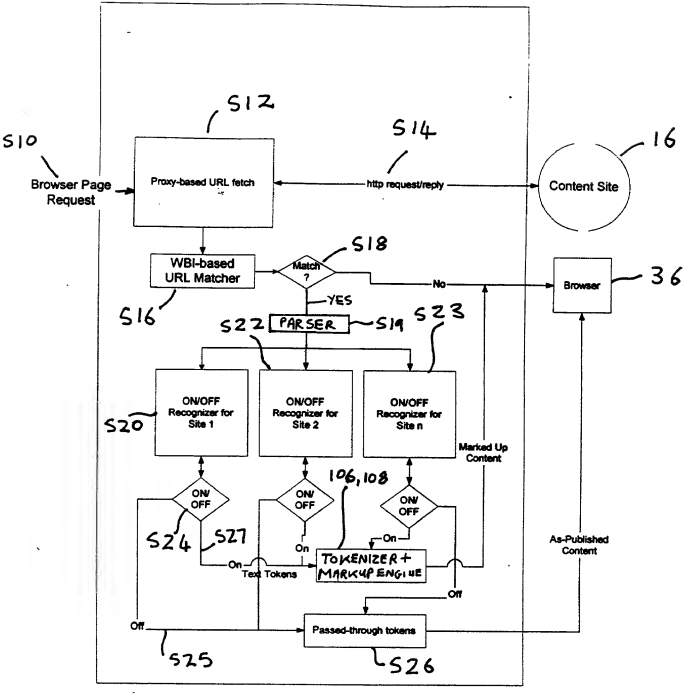


FIG. 5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Browser Page Request
S12
Proxy-based URL fetch
S14
http request/reply
16
Content Site
WBI-based URL Matcher
S16
Match ?
S18
No
YES
Browser
36
PARSER
S19
S22
S23
ON/OFF Recognizer for Site 1
ON/OFF Recognizer for Site 2
ON/OFF Recognizer for Site n
S20
Marked Up Content
106,108
ON/ OFF
S24
S27
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Text Tokens
TOKENIZER + MARKUP ENGINE
As-Published Content
Passed-through tokens
S25
S26

FIG. 6

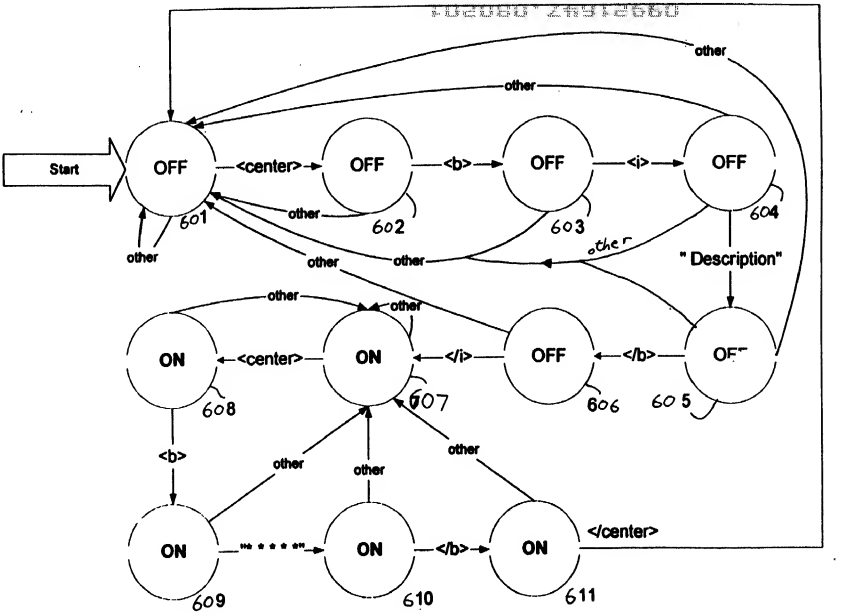
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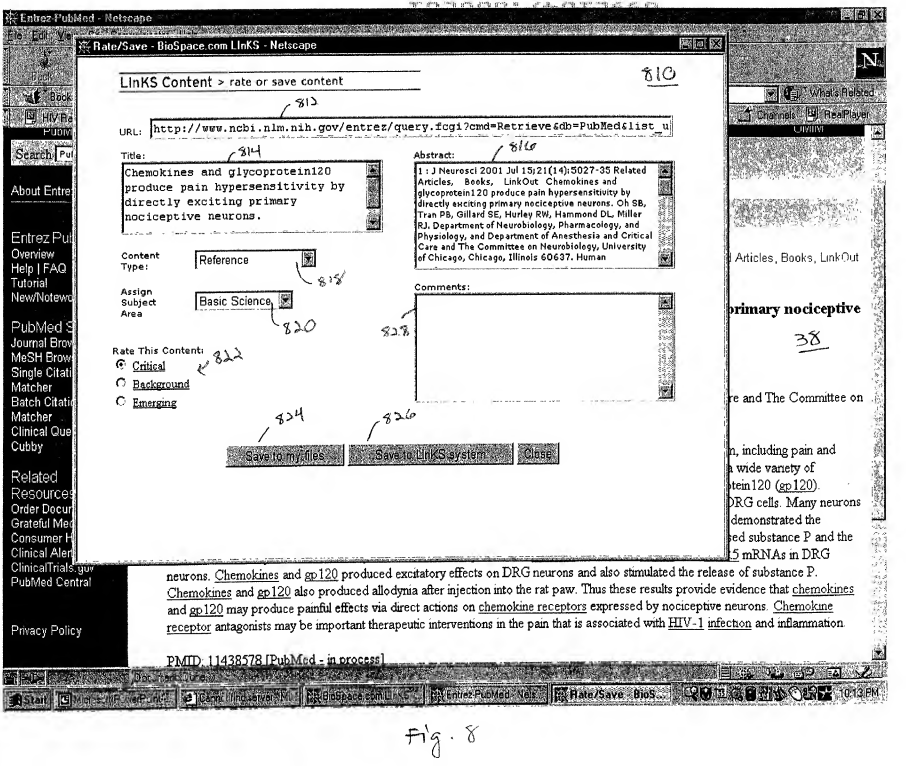
FIG. 7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814
Title:
Chemokines and glycoprotein120 produce pain hypersensitivity by directly exciting primary nociceptive neurons.
816
Abstract:
1: J Neurosci 2001 Jul 15;21(14):5027-35 Related Articles, Books, LinkOut Chemokines and glycoprotein120 produce pain hypersensitivity by directly exciting primary nociceptive neurons. Oh SB, Tran PB, Gillard SE, Hurley RW, Hammond DL, Miller RJ. Department of Neurobiology, Pharmacology, and Physiology, and Department of Anesthesia and Critical Care and The Committee on Neurobiology, University of Chicago, Chicago, Illinois 60637. Human
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818
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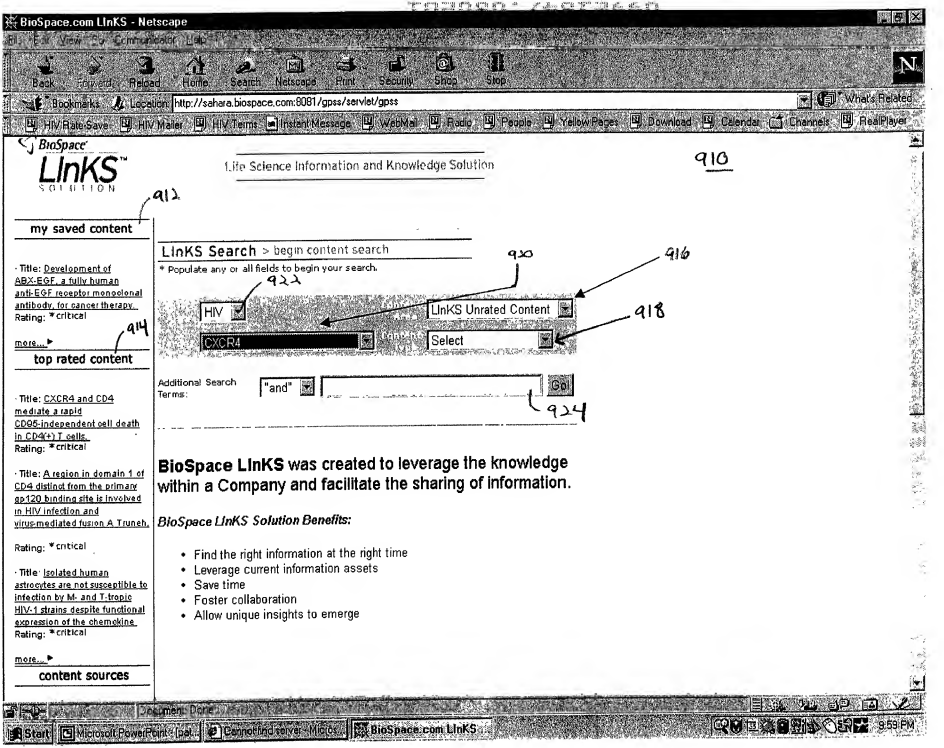
Fig. 8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· Title: Isolated human astrocytes are not susceptible to infection by M- and T-tropic HIV-1 strains despite functional expression of the chemokine
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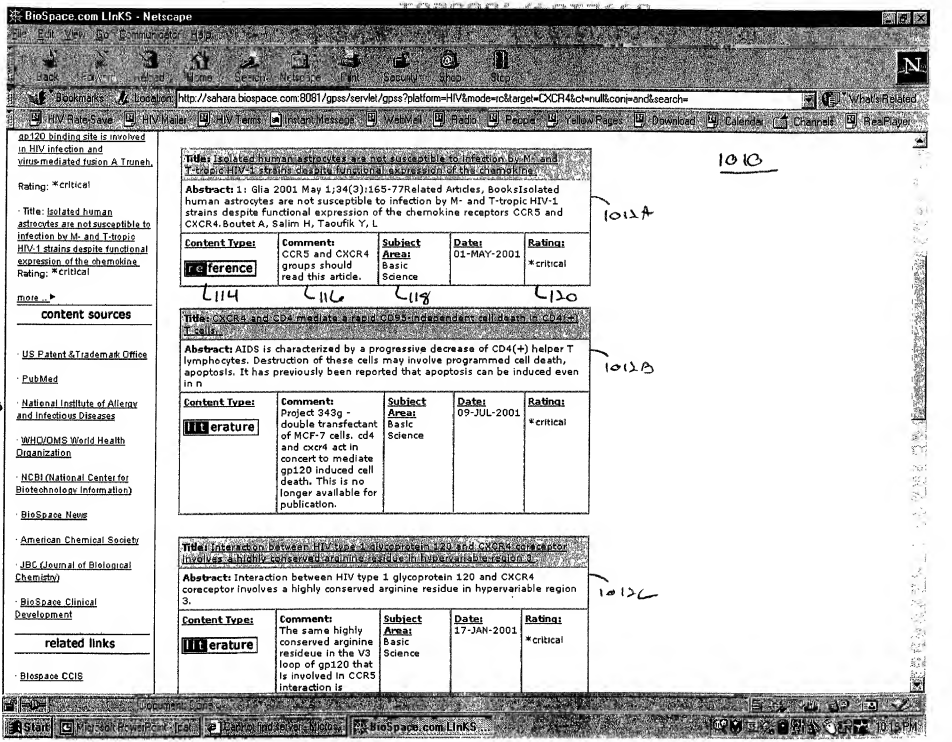
Fig. 9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Title: Isolated human astrocytes are not susceptible to infection by M- and T-tropic HIV-1 strains despite functional expression of the chemokine ...
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Comment: CCR5 and CXCR4 groups should read this article.
Subject Area: Basic Science
Date: 01-MAY-2001
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Abstract: AIDS is characterized by a progressive decreasa of CD4(+) helper T lymphocytes. Destruction of these cells may involve programmed cell death, apoptosis. It has previously been reported that apoptosis can be induced even in n
Content Type: literature
Comment: Project 343g - double transfectant of MCF-7 cells. cd4 and cxcr4 act in concert to mediate gp120 induced cell death. This is no longer available for publication.
Subject Area: Basic Science
Date: 09-JUL-2001
Rating: *critical
Title: Interaction between HIV type 1 glycoprotein 120 and CXCR4 coreceptor involves a highly conserved arginine residue in hypervariable region 3.
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Content Type: literature
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Subject Area: Basic Science
Date: 17-JAN-2001
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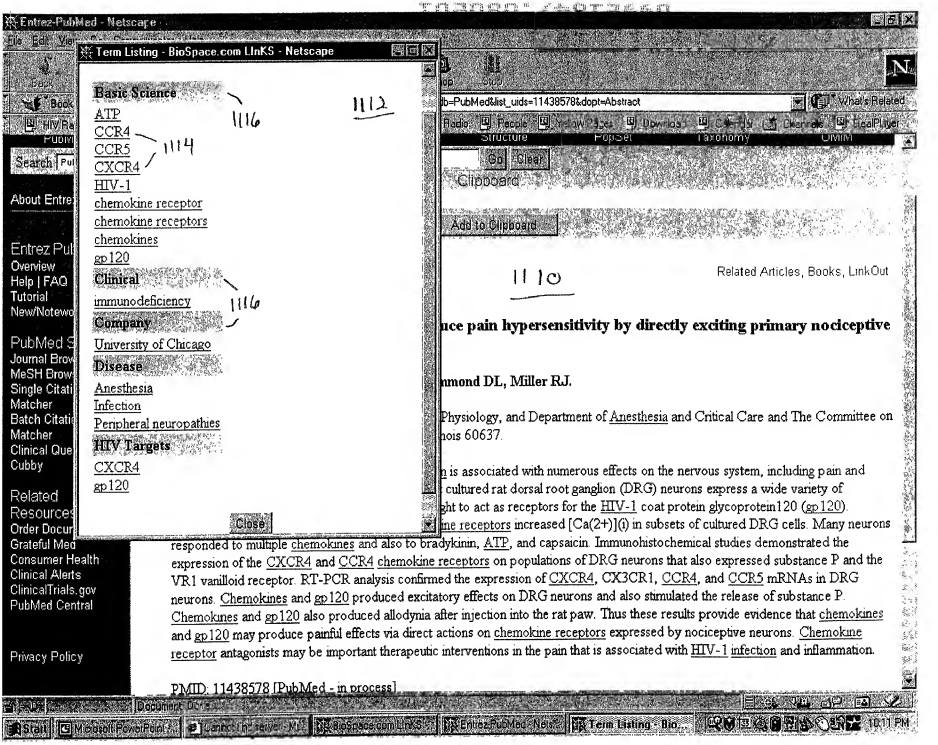
Fig. 10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CCR5
CXCR4
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chemokine receptors
chemokines
gp120
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n is associated with numerous effects on the nervous system, including pain and
cultured rat dorsal root ganglion (DRG) neurons express a wide variety of
ght to act as receptors for the HIV-1 coat protein glycoprotein120 (gp120).
ne receptors increased [Ca(2+)](i) in subsets of cultured DRG cells. Many neurons
responded to multiple chemokines and also to bradykinin, ATP, and capsaicin. Immunohistochemical studies demonstrated the
expression of the CXCR4 and CCR4 chemokine receptors on populations of DRG neurons that also expressed substance P and the
VR1 vanilloid receptor. RT-PCR analysis confirmed the expression of CXCR4, CX3CR1, CCR4, and CCR5 mRNAs in DRG
neurons. Chemokines and gp120 produced excitatory effects on DRG neurons and also stimulated the release of substance P.
Chemokines and gp120 also produced allodynia after injection into the rat paw. Thus these results provide evidence that chemokines
and gp120 may produce painful effects via direct actions on chemokine receptors expressed by nociceptive neurons. Chemokine
receptor antagonists may be important therapeutic interventions in the pain that is associated with HIV-1 infection and inflammation.
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Fig. 11